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OCTOBER, 1958

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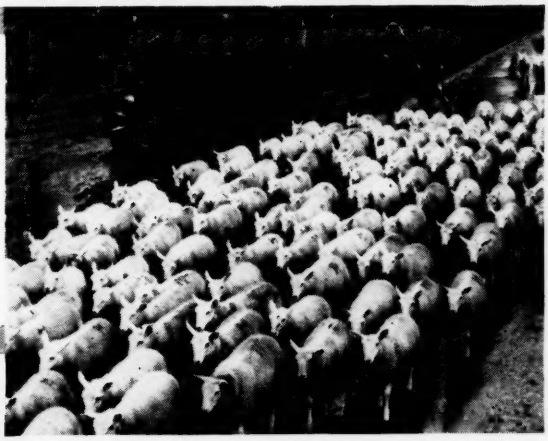

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EDITORIAL

AVETE

By the time that this journal is in the hands of our readers a new session has begun in the several veterinary schools and more than 200 young men and women will have embarked upon a student career in full hope that they may become veterinary surgeons.

Of what manner and regard are these aspirants. Some will be impelled upon the adventure by a strong vocational urge, some will have been influenced by environment and family association, others will come in search of a means of livelihood with no particular attraction to a career in veterinary science and indeed they would be perfectly happy in any other job which gave promise of a commensurate financial reward. Whatever the motive which caused them to pass through the portals of a veterinary school all have one thing in common. They will have negotiated with varying degrees of success a preliminary educational test designed to assess an adolescent's mental ability and competence and in addition they will have borne their witness before a selection board whose members are supposed to be competent to pass judgment upon human intellectual merit. Having survived the hazards of these obstacles it might be presumed that the fortunate students who gained entry to the course of training would not find the curriculum unduly exhausting nor the various examination tests very difficult. Unfortunately such a presumption has no basis in fact and only a relatively small percentage of the students complete the course in the prescribed minimal period. These statements apply equally to students in all disciplines but it would appear that the casualties in the medical and veterinary schools are registered at a higher rate than is witnessed in other spheres and the graph rises to the highest point in the veterinary schools.

Until recent times it was possible for any boy or girl to proceed to the course leading to a medical or veterinary qualification provided that they had attained to the minimal standards of the preliminary educational test and were able to arrange for

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the necessary financial support. There were more places than candidates and as the schools depended in no small measure upon student fees for sustenance a candidate applying for admission to the portals was seldom refused. When the number of candidates began to exceed the number of places some degree of selection became necessary.

Entry by selection was welcomed in academic circles. It was thought that the standards of intelligence and diligence would be enhanced and consequently the final product would be fitted better to enter a learned profession than his forebears had been. As the competition for places became more acute the selection standards were raised and a candidate who only possessed the minimal pre-entry requirements had little chance of entering upon the career of his choice.

All these things came to pass but the results have not been up to the hopeful expectation of those who welcomed selection and in many cases they have been worse than they were in the past when there were no restrictions on entry.

It may be that the methods of the selectors have been at fault for, in theory, selected material should be of better quality than that which has not been subjected to test. It is interesting to ponder upon the qualities that should be manifested by one who desires to enter a profession and especially a profession devoted to the healing art. In the main these requirements can be listed under three headings, educational background, vocational urge and personality.

It may be that too much emphasis has been placed on educational attainment as judged by the number of subjects specified on the matriculation certificate. The number of subjects does not indicate any capacity other than that it is a record that the candidate is possessed of a good temporary memory or that he has studied under men who "know the way to success in examinations". A youth in possession of a "good certificate" at the age of 17 years is not necessarily more intelligent than his fellows who are registered as being of lesser calibre. If we suppose that he is of a higher calibre at the age of 17 years it does not mean that this superiority will continue in the succeeding years and this fact is brought to notice constantly.

All candidates must be able to give evidence of a reasonable basic education but the factors which are of most importance in a professional life are vocational urge and personality. Many students in possession of high grade educational certificates are devoid of any vocational urge. They would be happy in any job which assured them of a reasonable standard of life. On the other hand there are many not so well placed on the educational scale who are impelled by a true vocational urge to give service in a chosen career yet are denied the chance to prove their worth.

It may be that if more emphasis were placed by the selectors on vocational urge, personality and the desire to give service the hopes of those who welcomed the selective method of entry would be implemented.

THE SIMPLEST SLAUGHTER FACILITIES

by

Dr. I. MANN

Department of Veterinary Services, Kabete, Kenya.

Field Slaughtering*

In many places where there are no public slaughter-houses, butchers are permitted to slaughter animals wherever they choose: an empty room in a dwelling-house, a backyard in a densely-populated area, the branch of a tree or a thicket in a banana grove, may be used for this purpose. Often the condition of the premises is quite indescribable and yet many of them exist not only in remote areas, but on the fringes of large towns and in small townships, with a population sufficient to justify a public slaughter-house. Such "slaughter-houses" are tolerated near schools, churches and cinemas, behind latrines and in other unsuitable locations. The meat is sold on the spot, and also in the towns. The primitive conditions often result from a misinterpretation of the law to the effect that once a butcher is in possession of a valid licence he may carry out his trade, i.e. slaughtering, wherever it is convenient for him to do so.

Meat which comes from these places is highly contaminated, has low keeping qualities, is not inspected, and often it is of grave danger to public health because it may be contaminated with human excreta.

The unfenced slaughter site may become a focus and reservoir of infectious and parasitic diseases due to the slaughter of sick animals. Uncontrolled slaughtering places offer an easy means of disposing of stolen livestock, and animals may be slaughtered for sale while in a moribund state because of disease, parturition, or accident.

These unhygienic and dangerous practices can be resolved by the erection and licensing of slaughter slabs.

Slaughter Slabs and Primitive Slaughter-houses

In remote and sparsely populated areas only, where the consumption of meat is very small and where beasts are slaughtered occasionally, the local authorities or the butcher should erect a slaughter slab.

The basic difference between a slaughter slab and the smallest slaughter-house is that a slaughter-house may consist of one or several slaughter slabs under a roof, and be enclosed by a wall, whilst the slaughter slab has neither roof nor walls.

Even if they are small in size, slaughter-houses or slaughter slabs should be provided with certain basic facilities for humane slaughter, and for the maintenance of a fair standard of hygiene and meat inspection.

The main requirements are as follows. The slaughter place should:—

(1) not be part of a dwelling-house;
(2) not be on a road leading to a dwelling-house;

* This article is a chapter from a book entitled "Meat Handling in Underdeveloped Territories, commissioned by the Food and Agriculture Organisation of the United Nations, Rome."

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(3) be at a reasonable distance from human habitation so that it may not become a nuisance to the neighbourhood;
(4) not be near latrines;
(5) be screened from view;
(6) have a hard, smooth, impervious flooring, sloping towards a drain and grooved to give better footing to beasts and staff;
(7) be provided with a wooden or iron frame, not less than 12ft. above floor level, with hoisting facilities;
(8) be provided with one or more floor rings to secure the animals;
(9) be provided with a water point on the slaughter slab, or a hose-pipe of sufficient length to connect to the nearest water point if this is not more than 25ft. away;
(10) have adequate facilities for the disposal of effluents;
(11) be provided with meat and offal hanging rails;
(12) have a scalding vat for pigs;
(13) have electric or pressure lamps to give adequate light to both butcher and inspector if slaughtering takes place at night;
(14) be properly fenced to prevent the access of other animals, or unauthorised persons.

Generally speaking, the code of conditions should protect the animal from undue cruelty, the neighbours from nuisance and the consumer from meat unhygienically slaughtered and handled which may be sold for consumption without being examined by a competent inspector.

Wherever possible, properly constructed slaughter-houses are best, for they offer screened working conditions and protection from rain.

Design and Equipment

A basic factor in hygienic slaughtering and producing meat of good keeping quality, is the lifting of the carcase. The most primitive form of hoist is to lift the carcase by means of ropes thrown over a horizontal beam 12ft. high. Owing to the effort involved, this method is justifiable only when the occasional beast is slaughtered.

No matter how small a slaughter slab or house may be, it must be equipped with proper lifting facilities which can be operated by one or two men. There are three methods which may be applied.

(*a*) *Two sets of blocks and tackle* arranged so as to lift and pull the sides apart to facilitate splitting.

(*b*) *Chain block and gambrel.* The chain block lifts the carcase and it can be fitted so that it may be used to convey the carcase as well. The gambrel or beef tree parts the sides.

(*c*) *Winch or wall hoist.* The winch lifts the carcase, and, as before, the gambrel parts the sides. If used in a slaughter-house provided with an overhead rail the carcase can be moved on to roller hooks.

A tubular gantry hoist is particularly suitable for countries where good quality timber is not available. It is suitable for use in those places where a fair number of large and small animals are slaughtered and the meat removed at once. When funds permit and the climate requires it, the gantry hoist should be protected by a roof as shewn in Fig. 3. The details of the concrete slab required for the gantry hoist when erected on a permanent site are shewn.

The slaughter slabs which already exist without hoist facilities could be improved by installing such a gantry. This would ensure that the carcase is lifted from the contaminated floor. Uniform equipment would allow for the interchanging of spare parts and the training of butchers on a country-wide scale.

If good timber can be secured the hoists etc. can be built of wood.

Where pigs are slaughtered a scalding tank is required. The tank can also be used to provide hot water for washing down.

There is no basic difference between slaughter slab and small slaughter-house, except that the latter is covered and enclosed. Although the plan shows a hoisting arrangement for two carcases on one slab, this has been done in order to ensure the greatest economy in space. For optimum working conditions the slab should be enlarged by two feet or should carry only one set of hoists.

Stage 3 represents a further development by providing two separate halls for cattle and sheep and goats, an optional extra hall for pigs, a tripery and a store. This layout is preferable for a small rural community where individual butchers pay a fee and slaughter and dress their own carcases. They dislike using a common hall because of the risk of confusion and dispute over the ownership of parts of the carcase.

Although this segregation is never recommended it may be essential if scattered butchers are to be brought together for control and improved sanitation. In time when the communal feeling has had opportunities to develop, a single-floor modern type slaughter-house can be adopted.

Any number of halls may be built but it is advisable to induce a number of butchers to use one hall so that for economic reasons six head of cattle and/or ten head of small stock can be killed in each hall each day.

If no pigs are slaughtered the pig slaughter hall may be omitted or converted to a cattle hall.

Licensing

Slaughter slabs or houses should not be privately owned, especially in under-developed territories, as the hygienic standards rapidly deteriorate without a constant battle between the butchers and the sanitary authorities. Every endeavour should be made to ensure that they are owned by the local authority which controls licensing, collects slaughter fees and is responsible for sanitary control.

When publicly owned slaughter-houses cannot be provided, a butcher's co-operative or several butchers who have pooled their funds may be allowed to erect a slaughter-house for joint use. Caution in these matters is necessary so that the licensing of privately owned slaughter-houses will not be found to be an obstacle in the way of providing public slaughter-houses, because the owners of these established premises will require compensation.

In order to be able to control such an establishment efficiently, a dual licensing system is required, one for the butcher and one for the premises. This enables the premises to be licensed after they have been inspected and approved by the local authority.

Both the granting of a new licence and the renewal of an existing one may be refused either if the applicant is considered unfit or the premises are considered unsuitable. Legislation should be enacted making it an offence to use premises for

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slaughtering purposes unless the butcher holds a valid licence in respect of these premises.

Licences should be granted for a period of not more than one year. A Health Officer's written report to the effect that both premises and equipment were inspected and found to be in good working order and that sanitary conditions have not deteriorated since the previous inspection, should be conditional to the renewal of a licence.

Whenever Meat Inspection services can be provided, the exact days and times at which slaughtering may be performed should be stated in the licence, so that the Inspector may be available for ante- and post-mortem examinations.

If in a Local Authority's opinion the number of private slaughter-houses operating in their district is sufficient, all future applications for additional private slaughter-houses should be refused.

The butcher or butchers using a private slaughter-house must be in possession of valid butcher's licences, similar to those held by butchers using public slaughter-houses. The granting of such a licence should depend upon whether, in the Local Authority's opinion, the butcher is a fit person to practise his trade and whether his services are required in this district.

Thanks are due to the Director of Veterinary Services, Kenya Colony, for permission to publish this article.

HISTOCHEMICAL INVESTIGATIONS ON THE RENAL FUNCTION IN THE COW FOETUS

by

S. TALANTI

Department of Anatomy, Veterinary College, Helsinki, Finland.

The metanephros, which is the actual mammal kidney, develops at an early stage of the embryonal development. It is known that the renal function begins during foetal life. Zietschmann & Krölling (1955) state that this function begins in the latter half of pregnancy. The renal function of the foetus has been investigated by various methods (Windle 1940), but a few investigations have been carried out in which histochemical methods have been used.

Primarily renal function can be investigated histochemically by demonstrating the components of urine in the tissue of the kidney. The typical components of urine are urea and uric acid, and in theory an investigation appertaining to these substances should give one some idea whether the kidney functions in a normal manner or not. It was realized, in preliminary investigations, that the methods employed for demonstrating urea were not sufficiently specific nor did they give a precise conclusion as to the localization of urea in the tissue. On the other hand the methods used for

demonstrating uric acid gave a sufficiently accurate result. The urine of domestic animals, including the cow, is relatively deficient in uric acid (Dukes 1943) but this fact does not present difficulties sufficient to hamper the investigations. Kinnunen & Paalanen (1952) have used the demonstration of uric acid as an indicator of secretion of urine in the human kidney.

The purpose of the present study was to investigate the renal function of the cow foetus. Particular efforts were made to ascertain the stage at which the first evidences of uric acid occurred in the tissue of the kidney.

Material and Methods

Material consisted of 23 foetuses of healthy cows. They were removed from the uteri immediately after the cows were killed. The kidneys of the foetuses were removed, cut into pieces and fixed in absolute alcohol for at least 36 hours. The pieces were embedded in paraffin and cut sagittally at 5 to 15 micra. Staining for uric acid was done according to Schultz & Schmidt's method (1931). In some instances the original method was followed, in others the counter-staining was omitted so that a more distinct picture could be produced.

The age of the foetuses could not be ascertained; it was estimated by using the table of Richter & Götze (1950). According to it the calculation of age is based upon the body length. The body lengths of the foetuses were 10, 10, 12, 14, 17, 18, 19, 20, 22, 24, 25, 27, 29, 30, 36, 44, 49, 50, 64, 74, 77, 80 and 83 cm., the estimated ages 3, 3, 3, 4, 4, 4, 4, 4, 4, 4, 5, 5, 5, 5, 6, 6, 7, 7, 7, 7, 8, 8 and 8 months respectively.

Results

Uric acid was found in the tissue of the kidney in all the foetuses investigated. It was demonstrated as crystals and mainly in the tubules (Fig. 1 and 2). In the youngest foetuses crystals appeared only here and there. Judged by the eye the amount of uric acid seemed to increase as the foetal age advanced. Investigations made for the sake of comparison showed that the amount of uric acid in the tissue of the kidney of the oldest foetuses was less than in that of newborn animals.

Discussion

The results suggest that the renal function of the cow during foetal life begins at an earlier stage than some investigators have supposed. Kinnunen & Paalanen (1952) achieved similar results with regard to the human foetus.

One of the functions of the kidney during foetal life is to participate in producing foetal fluid (Shaw & Marriot 1949). The allantoic and amniotic membranes produce foetal fluid also. The quantitative share in this process is not known. According to the earliest conceptions the renal function produced most of the foetal fluid in man and the allantoic fluid in some animals was regarded as mere foetal urine (Richter & Götze 1950). Some investigators have claimed that the renal function is of little or minimal importance in the producing of foetal fluid (Tausch 1936, Fitschen 1941). As the foetal fluid contains components similar to urine, it seems logical to draw the

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conclusion that the renal function plays a part in its production. The fact that the renal function begins at an early stage of gestation gives some support to this opinion. The continual increase of the urine component in foetal fluid (Makepeace *et al.* 1931) suggests that the renal function continues throughout pregnancy. This view is in line with the results obtained in this investigation. Although the method used was not quantitative, the increase of uric acid in the tissue of the kidney was so positive during the advancing foetal development that it suggests that the intensity of the renal function increases continually although the secretion does not rise to a post-natal level. The results concerning the amount of allantoic fluid in the foetus of the cow (Arthur 1957) also indicate that the renal function increases continually during foetal life.

It is interesting to compare the development of the hypothalamic-hypophysial neurosecretory system regulating the renal function with the results obtained here. It was observed in previous investigations that this system develops at a very early foetal stage (Kivalo & Talanti 1957). Stainable neurosecretory material could be demonstrated in this system in the cow foetus at the early age of three months. According to the present conception the principles of the posterior lobe of the hypophysis appear in this material (Bargmann 1956). Thus the antidiuretic factor controlling the renal function can be demonstrated in the hypophysis even at this early stage when, according to this investigation, renal function is found to exist.

Summary

An attempt was made to investigate the renal function in the cow foetus. The method used was calculated to demonstrate uric acid histochemically in the tissue of the kidney. Crystals of uric acid were demonstrated in the kidneys of the foetuses at an early stage when the body length of the youngest foetuses was 10 cm. The results are discussed in the light of those achieved previously in the investigations concerning the production of foetal fluid and the development of the hypothalamic-hypophysial neurosecretory system.

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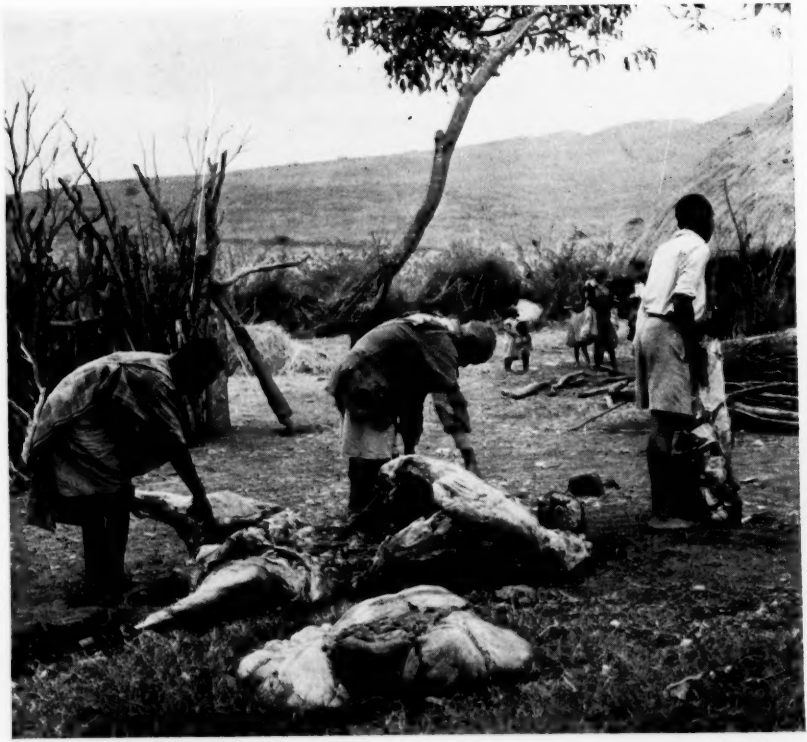

FIG. 1
Slaughtering in the Field

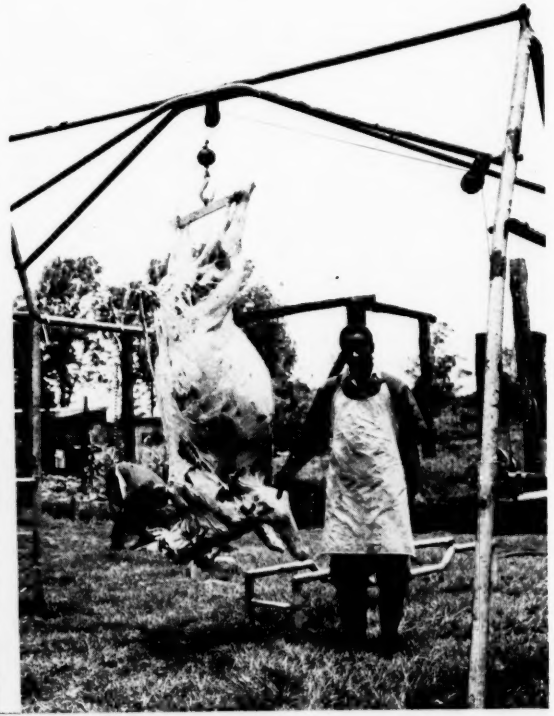

FIG. 2
Simple Hoist

PLATE 1

(Article by Dr. I. Mann on pages 371-374)

PLATE 2

FIG. 3
A roofed, double hoist at an Agricultural Exhibition

(Article by Do. I. Mann on pages 371-374)

THE USE OF HEXADECAMETHYLENE-1:16 BIS (ISOQUINOLINIUM CHLORIDE) IN THE TREATMENT OF RINGWORM IN DOMESTIC ANIMALS

by

T. N. GOLD, M.R.C.V.S. (*Redditch, Worc.*)

and

BRUCE V. JONES, M.R.C.V.S. (*Clavering, Essex*)

Introduction

Recent work on veterinary mycology, particularly that concerned with the dermatophytes, has pointed to the existence of a reservoir of ringworm infection in the animal population in Great Britain.

Some measure of the importance that is attached to animal ringworm can be gathered from the reports of the Technical Development Committee of the B.V.A. (1955) and the M.R.C.'s Medical Mycology Committee (1956). As well as these the results of a survey of animal mycoses in Great Britain by the Agricultural Research Council (Ainsworth and Austwick, 1955), and the publication of clinical papers by Mortimer (1955) and La Touche (1955) indicate also the importance of this animal reservoir.

Of the farm animals ringworm occurs most frequently in cattle and equine ringworm is not uncommon. In pigs it appears to be rare in Great Britain and of little, if any, significance.

(*a*) *Ringworm in cattle*

In cattle the occurrence of ringworm has serious consequences which can be discussed under two headings: (a) the economic loss to the cattle industry, and (b) transmission of ringworm to man.

The economic loss to the cattle industry is difficult to calculate because little information is available on the incidence of the disease. During the winter months the incidence rises considerably, particularly in young stock. (This rise might be artificial since in these months the animals are housed and under more critical observation). La Touche (1955) records that the infection rate for cattle affected with *Trichophyton discoides* is very high and that on occasional farms it is not unusual to find all calves infected. Sellers (1956) has reported that in Yorkshire *T. discoides* infection is found on a high proportion of farms, being seen most frequently on calves in their first year of life and during the winter. Ainsworth and Austwick (1955) in reporting the results of the A.R.C. survey found that 50% of the confirmed ringworm samples were from cattle, all being *T. verrucosum* var *discoides*. The limited data collected in this survey suggested that the incidence showed little seasonal variation, and it is thus at variance with the commonly accepted views. Mortimer (1955) in a survey of approximately 2,000 cattle on infected premises in East Anglia reported an incidence of 15%. He noted also that ringworm was prominently a disease of young stock and beef cattle.

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Control of cattle ringworm is necessary because of its importance as a source of human infection, but it also possesses some significance as a disease per se. Although, as a rule, cattle lesions appear to be of a superficial nature, and manifested only by loss of hair and crust formation, it has been reported that infection may become extensive and severe in some animals (Georg, 1954). Reduction in milk yield and bodyweight associated with *T. discoides* infection in dairy cows in Canada has been reported (Blank, 1953). Mortimer (1955) noted that, although the lesions are not in themselves painful, when they are extensive they appear to cause some pruritis, and the irritation causes a delay in healing and encourages the creation of new lesions. He noted that in severely infected calves there was evidence of retarded growth. Kral (1955) stated that advanced cases in young animals in poor general condition sometimes take a severe course. One cannot be certain whether the animals loose condition as a result of the infection or vice-versa. This was the position in an outbreak reported by Brander (1955) in which cattle with *T. discoides* infection were found to be in extremely poor condition, the lesions including extensive raw areas.

Kral and Novak (1953) have described the histopathology of the lesions and noted that folliculitis and suppuration develop frequently. They state that widespread infection may interfere with nutrition. Sellers *et al* (1956) have reported their observations on the histopathology of the natural and experimental disease in cattle and observed a marked inflammatory response to the invasion of the parasite. This becomes more widespread with the spread of the infection and in due course, intra-dermal and intra-epidermal micro-abscesses form.

The transmission of bovine ringworm to man can result in a troublesome and distressing disease due to the production of kerions and tinea circinata (Martin-Scott, 1955). Recently the importance of this bovine reservoir in the production of human cases has been emphasised by reports in medical journals.

(*b*) *Ringworm of horses*

It has been assumed that ringworm in the horse has been rare in recent years but it would appear to be not uncommon, at the present time, in establishments where numbers of horses are kept, such as breeding, riding or training stables. In the past there has been some confusion regarding the nomenclature and multiplicity of the dermatophytes isolated. This latter point has been clarified to some extent by the results of the survey by Ainsworth and Austwick (1955) in which *Microsporum equinum, Trichophyton discoides* and *Trichophyton equinum* were isolated from equine samples, the latter being observed most frequently.

The apparent "rareness" of the condition may be due to the fact that the owner is liable to a considerable fine if a horse with ringworm appears on a race track. This may explain the colloquial names that have been given to this condition such as "girth itch" and "Canadian horse pox".

The contagiousness of the disease is marked and usually a characteristic rapid spread is seen following the introduction of an infected animal to a previously clean stable. Common causes of transmission or the introduction of infection have, in our experience, been travelling in infected horse boxes, transmission via infected saddles, harness or grooming equipment, breeches or boots of riders and also the practice of "race-cloths" being supplied by the track and being used on many horses in succession.

The disease causes considerable discomfort to the animals which cannot be used for riding while infected, and racing, breeding, or riding schedules may be interrupted seriously.

(*c*) *Ringworm of pigs*

Ringworm in the pig appears to be very rare and very few incidents have been recorded in Great Britain. A recent report (McPherson, 1956) has recorded the isolation of *T. mentagrophytes* from pigs. The disease was self-limiting and healed in less than 10 weeks without treatment. McPherson drew attention also to the condition *Pityriasis rosea* in pigs which is often erroneously termed " ringworm ".

Treatment

The treatment of ringworm has been the subject of much discussion and the recent comments by Mortimer (1955) and the Medical Mycology Committee (1956) indicate clearly that most of the available treatments are of doubtful value.

Because of these unsatisfactory results it was decided to undertake clinical investigations with a new anti-fungal preparation containing hexadecamethylene-1: 16-bis (isoquinolinium chloride). The pharmacology, antifungal and antibacterial spectrum of this compound has been reviewed by Forster (1957). Following the early reports of the use of other members of this series of compounds in the treatment of calf ringworm (Collier *et al*, 1955) a detailed report of the treatment of over 100 cases of bovine *T. discoides* infection has been published (Forster, 1957), in this report rapid and effective cures were reported in all cases. Further favourable reports on the clinical effect and efficacy of this compound in treating bovine ringworm have been published by Fowler (1957) and Crawshaw (1957).

Materials and Methods

The preparation used was a 0.25% solution of hexadecamethylene-1: 16-bis (isoquinolinium chloride)* in alcohol, also containing crystal violet (to act as a marker). (In some of the earlier cases treated a similar lotion was used without the crystal violet, this was found to give equally good results and was more satisfactory in the case of some thoroughbred horses as the clothing being used on these animals at the time was not stained by the dye). In later cases where a colourless solution was desired another preparation containing 0.4% of hexadecamethylene-1: 16-bis (isoquinolinium chloride)** was used, this product is available for the treatment of dermatophyte infections in man and was obtained for experimental investigation more easily.

These preparations were used in clinical practice for the treatment of ringworm in cattle, horses and pigs over a 30 month period. In all cases scrapings were obtained before or at the time of treatment and examined for the presence of hyphae and spores in a 10% potassium hydroxide solution. In almost all cases the clinical picture and history was characteristic. Laboratory examination was reserved for those cases where clinical examination and history were typical or strongly suggestive of a diagnosis of " ringworm " but where hyphae or spores could not be seen by direct microscopical examination. This was required most frequently in horse patients.

*Tinevet, Allen and Hanburys Limited.
**Teoquil Gel, Allen and Hanburys Limited.

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Results

(*a*) *Cattle*

During this period over 150 cases were treated. The majority of these occurred in Freisian, Hereford, Shorthorn and Jersey herds. Animals of all ages were treated but most of them fell within the 6-18 month old group. All cases presented the typical picture of *T. discoides* infection showing circular, crusted hairless lesions on the head, neck and trunk. This was confirmed by cultural examination in certain cases. At first the lesions were treated with daily applications of the lotion for one week and then once every third day during a second week. As experience accumulated this was reduced and equally good results with rapid regression were obtained by using an average of three applications at intervals of two to three days, some cases responding well after one application. Lesions were scraped or scrubbed before treatment to remove the crusts and the lotion was applied with a stiff brush. The stock-owners were advised to blow-lamp and creosote all buildings where these animals were housed. No signs of toxicity were observed even though many animals were observed to lick the lesions following treatment. Orbital lesions, even those completely surrounding the eyelids, were treated quite safely. The dye was most valuable in ensuring that all areas had been treated.

(*b*) *Horses*

In this series six outbreaks involving thirty-two horses have been treated. The numbers of animals involved in the six outbreaks treated were 5, 3, 6, 5, 3 and 10. In most cases other horses were present on the premises but spread to these horses was checked as far as possible by rigid sanitary controls and the rapid commencement of treatment. All of the animals treated in these outbreaks were thoroughbred horses. There was no specific age or sex incidence. In all cases scrapings were taken and examined microscopically for hyphae and spores. In three cases hyphae and spores were not found and samples were sent for cultural examination. *Trichophyton equinum* was isolated from two of these cultures, in the third it was not possible to isolate a dermatophyte. Because of the difficulty of isolating this fungus from equine samples, and the characteristic clinical picture presented by the outbreak and the response to treatment the authors have little doubt that the third outbreak was due to ringworm. Usually the lesions were in direct relationship to the position of the harness or saddle. One outbreak followed the transport of a horse in a box which had been occupied by a horse "with a skin disease", and others followed attendance at race tracks. The commonest sites of lesions were on the withers, neck and sternal region. During the outbreaks all grooming was suspended, all harness was scrubbed with disinfectant solutions and all clothing was sterilised; strict isolation and hygienic measures were introduced during the outbreaks to prevent spread of infection. The lotion was applied every day for a week by means of small camel-hair brushes. (Other paint-brushes, suitable for cattle, were found to be too severe for the thin skinned horses). Treatment was successful in all cases. Most animals were cured after one week's treatment, a small proportion required a longer period. When possible all buildings were disinfected, blow-lamped and creosoted before they were allowed to be used again.

The typical lesion observed, was of rounded elevated areas of various sizes, this was followed by loss of hair and frequently a red moist surface was left. Usually

the edge of the lesion was inflamed and showed many broken hairs. Itching was not present in all cases. Sometimes crusts formed and often coalescence of lesions was seen.

(*c*) *Pigs*

Only one outbreak was seen. Two sows and six gilts (Large Whites) were involved. These pigs were running in an open yard with a group of steers infected with *T. discoides*. The lesions took the form of diffuse patches which tended to occur on the back and fore limbs and to have crusted surfaces. Apart for some local irritation no other symptoms were observed, and the clinical appearance or signs did not correspond to that of *Pityriasis rosea*. Scrapings were taken and on examination spores and hyphae were seen in and around the hairs. The lotion was applied each day for one week. Complete resolution of the lesions occurred in this time. The infected cattle in the yard were treated also.

Discussion

The treatment of ringworm in farm animals presents many difficulties and in particular because resolution has been very slow and protracted. Control of outbreaks is difficult due to cross infection and the animal attendants have been at risk for a long period. The latter are often unwilling to handle the animals very frequently. Our observations on the use of a lotion containing 0.25% hexadecamethylene-1 : 16-bis (isoquinolinium chloride) confirm the results of other workers and indicate that this is an effective and rapid treatment for ringworm in cattle, horses and pigs. In the horse, in particular, it has produced very striking results, enabling a quick return to work and making the control of the very rapid cross-infection relatively easy. It has been impossible to conduct a controlled experiment within the limits of a general practice but the consistently successful results obtained, and agreement with other observations published on this compound, provide evidence of its therapeutic effect. In the treatment of cattle the addition of the colouring agent to the lotion is of particular value. During the course of these trials, two cases of ringworm occurred in men who handled the animals, both were treated and cured by the daily application of the lotion for one week.

Summary

The clinical trial of lotions containing hexadecamethylene-1 : 16-bis (isoquinolinium chloride) in the treatment of ringworm in cattle, horses and pigs is reported. From the outbreaks both *T. discoides* and *T. equinum* were isolated. Clinical cures were obtained rapidly in all cases treated. These results agree with those of other observers published on the treatment of cattle ringworm and would indicate the value of this antifungal compound in veterinary practice.

Acknowledgments

Our thanks are due to Mr. P. K. C. Austwick of the Ministry of Agriculture's Veterinary Laboratories for confirmation of the *T. equinum* infections and to Messrs. Allen and Hanburys Ltd. for the supply of the Tinevet Ringworm Lotion and Teoquil Gel used in these investigations.

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SIMPLE SCREENING TESTS FOR BLOOD SUGAR AND BLOOD UREA

by

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Introduction

Determinations of sugar (glucose) and of urea in blood are important steps in the diagnosis of certain metabolic diseases. Since estimations of these substances are carried out as a rule in a fully equipped laboratory it is desirable that simpler preliminary screening tests should be available. These would have many advantages, particularly in providing quick semi-quantitative answers.

It is the aim of this paper to describe modifications to two well known methods that enable them to be used in this manner.

Choice of Methods

The reduction of copper compounds figures largely in many of the colorimetric methods used for the estimation of glucose. None of these appeared to be particularly suitable, so attention was directed to Lee's modification (1954) of Sumner's original method using 3:5-dinitrosalicylic acid. This has been recommended as a rapid screening test for blood sugar in human diabetes mellitus etc., and makes use of permanent visual standards. The effective range is about 80-1400 mg/100 ml., with a practically linear relation between concentration and colour in the range up to 400 mg. 'Strong' colours, therefore, can be diluted to bring them within a range of suitable standards. The chief disadvantage of the method is that the colour of the blank is

yellow, and this is difficult to distinguish from colours resulting from glucose concentrations less than 80 mg./100 ml. (Fig. 1). Since 30-60 mg./100 is the normal range of blood glucose for sheep and for cattle, two methods have been devised for estimating them by Lee's method. These are described later in this communication.

Normally blood urea is determined by the urease method. Clearly this type of reaction is not suitable for 'field' use, therefore attention was directed to the Diacetyl Monoxime reaction described and reviewed by Natelson (1957). This reaction is not specific for urea (Ormbsy, 1942) and is given by other naturally occurring substances containing the carbamino group, e.g. citrulline. Since the normal concentrations of these substances in blood are low, and the colours resulting from their reaction with diacetyl monoxine less intense than that given by urea, they are unlikely to affect the final result in any semi-quantitative screening technique.

In selecting a suitable standard it was noted that aqueous ferric chloride had similar if not identical colours with those resulting from both reactions. Thus concentrations greater than 20 per cent. matched well with the glucose/dinitrosalicylic colour, and concentrations less than 10 per cent. matched reasonably well with the urea/diacetyl monoxime colour.

Equipment and Chemicals

1 ml. syringe	1 or more as required. The Tuberculin intradermal type fitted with small gauge needle.
Pipette, 1 ml., bacteriological type	As required. Syringes make excellent substitute pipettes for this type of screening test.
Pipette, 10 ml. in 1/10ths	One. A good 5 or 10 ml. syringe is equally satisfactory.
Glass funnels	2 or 3. About 1 in. diameter.
Filter papers	Whatman No. 1 or equivalent.
Test-tubes	Small size. Uniform diameter about 0.5 in. Capacity 10 ml.
Centrifuge tubes	Standard 15 ml. type, graduated in 1/10ths. These are suitable for carrying out the reactions and for subsequent dilutions in matching to the standards. They should be uniform in size and shape.
Sterile saline	As required.
Sulphosalicylic acid	6.0 g./100 ml. distilled water.
3:5-dinitro-salicylic acid	1.75 g./100 ml. Keep in slightly warm atmosphere to prevent crystallization.
Alkaline reducing agent	Phenol, 13.8 g., Sodium Bisulphite, 13.8 g., dissolved in caustic soda, 10 per cent. (w/v), 350 ml., and added to Rochelle Salt, 500 g., in water 880 ml.
Diacetyl Monoxime	2.0 g. in acetic acid (2 per cent v/v) 100 ml. Keep in amber glass bottle in the cold.
Acid Mixture	To water, (70 ml.) is added phosphoric acid (85 per cent, 75 ml.) and this is added *slowly* to sulphuric acid (concentrated, 25 ml.)
Ferric Chloride (100 per cent.)	100.0 g. of ferric chloride hexahydrate, (A.R.) dissolved in distilled water and made up to 100 ml.

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Other ferric chloride standards, e.g. 20 per cent. (100 mg. glucose/100 ml.), 10 per cent., and 2 per cent. (25 mg. urea/100 ml.) can be made by accurate dilution of the concentrated stock.

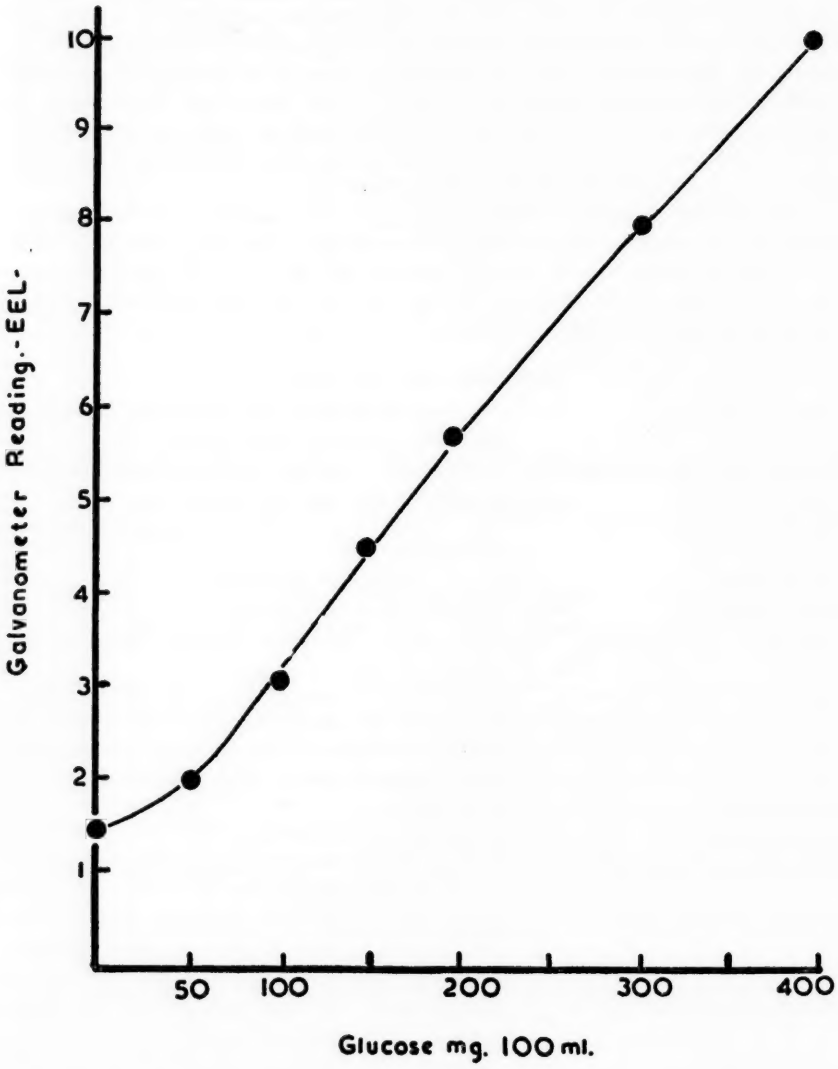

FIG. 1

Calibration curve of glucose; Lee's method. Quantities four times those quoted in the text, with saline in place of blood. Measurement made on EEL Colorimeter.

Collection and deproteinisation of the blood

The quantity of blood required for either estimation is 0.2 ml., or 0.4 ml. if a low blood sugar value is suspected (e.g. in hypoglycaemia or in normal ox blood). A good grade 1 ml. tuberculin intradermal syringe is filled to the 0.8 (or 0.6) mark with sterile saline. All air bubbles must be removed. 0.2 (or 0.4) ml. of blood is drawn into the syringe. This is inverted several times to mix, some of the diluted blood is expressed into the small test-tube and, after further mixing, the rest of the contents is added. Sulphosalicylic acid (1.0 ml.) is added to the diluted blood, the contents mixed well, allowed to stand 5-10 minutes and filtered through a small paper. About 0.8-0.9 ml. of a clear colourless filtrate is obtained. This is sufficient for a single glucose estimation and a single urea estimation, if 0.25 ml. or less is required (*vide infra*). It has been found that it is better to use 0.5 ml. of filtrate for both estimations *in the first instance*, and the following methods are based on this.

Development of Colours

An aliquot of filtrate is placed in a small calibrated test-tube, or in a graduated centrifuge tube and reagents are added as follows:

Glucose		*Urea*	
Blood Filtrate	0.5 ml.*	Blood Filtrate	0.5 ml.*
Dinitrosalicylic Acid ...	0.5 ml.	Diacetyl Monoxime ...	0.25ml.
Alkaline Reducing Agent	1.0 ml.	Acid Mixture	0.5 ml.

*If less than 0.5 ml. is used the volume is made up to 0.5 with saline, and a suitable adjustment made in the final calculation.

Colour Assessment

(1) *Dilution technique.* The principle of this technique is to dilute the unknowns until they match the appropriate standards. Since both standards are approximately equivalent to the normal levels of blood glucose and blood urea, any colours that are less intense than the standards can be taken to indicate a hypoglycaemia or a hypo-uraemia.

In practise it is suggested that known volumes of water are added in 0.5 or 1.0 ml. quantities to the reaction tubes until a reasonable match is obtained with the standards. The following dilutions have been worked out by matching colours from known concentrations of glucose and urea with dilute solutions of ferric chloride. The matching was carried out by means of a Duboscq colorimeter and also by eye with independent observers. Simple calibration/dilution curves are shown at Figs. 2 and 3.

Glucose Dilution Values

Amount of water added to 2.0 ml. of reaction mixture to match it with the 100 mg. glucose standard (ferric chloride 20 per cent.)	Indicating an original glucose concentration of:
ml.	*mg./100 ml.*
0	100
2.0	200
4.0	300
6.0	400

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Intermediate values may be obtained by use of the calibration curve (Fig. 2) and by adding smaller volumes of water.

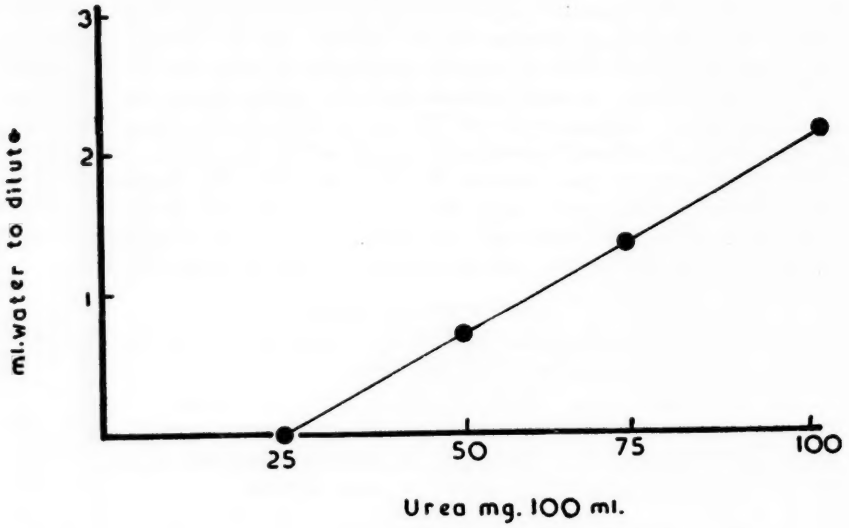

FIG. 2
Quantity of water added to 2.0 ml reaction mixture. Glucose/dinitro salicylic acid.

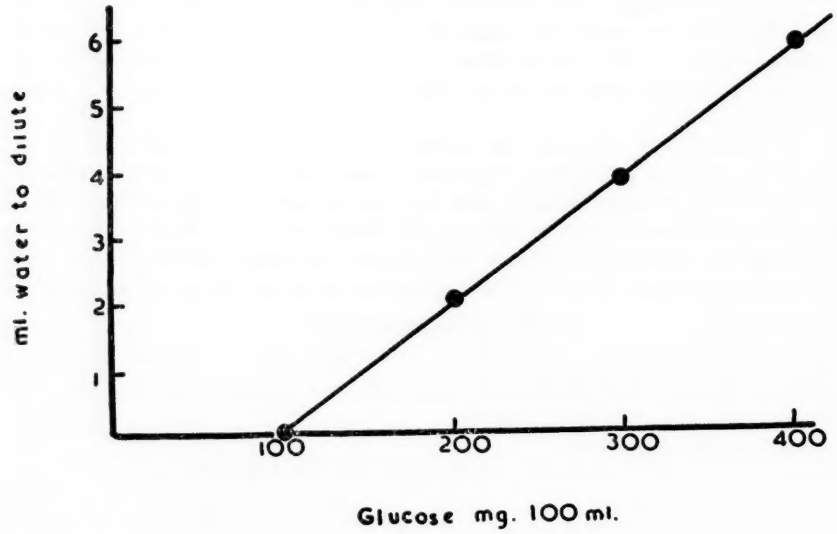

FIG. 3
Quantity of water added to 1.25 ml reaction mixture Urea/diacetyl monoxime.

Urea Dilution Values

Amount of water added to 1.25 ml. of reaction mixture to match it with the 25 mg. urea standard (ferric chloride 2.0 per cent.)	Indicating an original concentration of:
ml.	*mg./100 ml.*
0	25
0.7	50
1.5	75
2.2	100

It has been found that the calibration curve for diacetyl monoxime urea is not linear after about 100 mg./100 ml. In other words if on development the final colour suggests a urea concentration of 100 mg./100 ml. or more, a further estimation should be made on a much smaller volume of filtrate e.g. 0.1 ml. and diluting to 0.5 ml. with saline.

Alternative Method

Sets of permanent standards may be made by diluting concentrated ferric chloride (100 g./100 ml.) as shown below. These enable a match to be made without dilution of the unknown. These are less satisfactory for urea, where the colours are less easy to compare.

	Glucose: mg./100 ml.						
	400	300	250	200	150	100	50
ml. ferric chloride (100g./100 ml.) ...	9.0	8.0	6.7	5.3	3.3	2.0	1.3
ml. water	1.0	2.0	3.3	4.7	6.7	8.0	8.7
	Urea: mg./100 ml.						
			100	75	50	25	0
ml. ferric chloride (10 g./100 ml.)			4.0	3.7	2.6	1.5	0
ml. water			6.0	6.3	7.4	8.5	10

Procedure for estimation of glucose in ox blood

Preliminary investigations indicated that doubling the quantity of blood did not result in doubling the glucose concentration when estimations were made accurately by Lee's method using an EEL absorbtiometer. The explanation of this appears to be that values below 80 mg./100 ml. lie on the inaccurate part of the curve (see Fig. 1). Therefore half the concentration given by a double volume of blood is probably a more correct estimate. An alternative method is to add a known quantity of glucose to the sample, equivalent to 100 mg./100 ml., so that the resulting colour can be read on the accurate part of the scale. This method undoubtedly gives more accurate readings, but it is suggested that for ordinary screening techniques a double volume of blood—without added glucose—will give adequate results.

Some recoveries of glucose from the blood of two steers are given in Table I.

These figures illustrate the points previously made and emphasise that by using the accurate part of the calibration curve the recoveries are good, and show little difference whether based on single or half double values.

Summary

Two simple techniques are described for the rapid estimations of blood sugar and blood urea. Either estimation can be carried out within 20 minutes, and values

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Table 1

Recovery of glucose from the blood of two steers, A and B.
(EEL Absorbtometer, Green, OGRI Filter)

Sample	Glucose added (mg/100 ml.)	Volume of blood taken	Glucose mg/100 ml.			Recoveries: per cent. on:—	
			Duplicates		Mean	Single Volume	Half Double Volume
A	—	1	67	60	65	—	124
A	—	2	100	110	105	78	—
B	—	1	30	20	25	—	119
B	—	2	35	50	42	84	—
A	50	1	108	110	109	95	106
B	50	1	82	78	80	107	112
A	100	1	165	170	167	101	108
A	200	1	270	280	275	104	108
		Average Recoveries +/— s. d.				95 +/—10.5	114 +/— 6.0

± 25 mg. can be obtained in the case of glucose, and ± 12.5 mg. for urea. Lee's method can be adapted to the estimation of very low blood sugar levels on a quantitative or semi-quantitative basis.

One of the main advantages of the methods described is the use of permanent ferric chloride standards. These are suitable for either the dilution or straight comparison techniques more particularly for glucose. For most purposes the first method is adequate in which case two standards only are necessary, namely, 20 per cent. ferric chloride for glucose and 2 per cent. for urea.

The equipment and chemicals are inexpensive and readily obtainable, and the amounts described are suitable for about 200 estimations.

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CANINE SPIROCERCOSIS WITH SPECIAL REFERENCE TO THE INCIDENCE AND LESIONS

by

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The worm *Spirocerca lupi* infects dogs, foxes, wolves and jackals and the infection is prevalent in many countries. Neumann (1892) stated that the worm was first detected in aortic 'tumours' of dogs by Morgagni and Courten in the year 1760. Later Manson (1888) in China, and Lewis (1888) in India reported the occurrence of the same condition. The earlier workers mentioned that besides the localisation of the worms in the oesophagus and arterial wall, they could occasionally be noticed in the lumen of the aorta where they produced thrombosis and rupture, and also in the

lymphatics in the vicinity of the oesophagus. More recently, some general accounts of the lesions have been given by Oro (1931), Leivate (1933), Sanchez Boligo (1936), Mundhenk and Greene (1939), and De Cross (1941). Data on the incidence of spirocercosis have been furnished by Krameveld & Djeaneodin (1952) in Sumatra, Barron (1950), Rubin (1952), Guberlet (1923) and False and Newberne (1954) in the United States of America.

In this country, though it is common knowledge that the disease is present in a clinical and non-clinical fashion, no data have been published to furnish information on the extent of infection. To make good this deficiency, an analysis is made of the large volume of information available in the Department of Pathology of this College. Incidentally, the study provides an opportunity for noting the location and nature of the lesions in a large number of cases included in this survey.

Materials and methods

Autopsy records for a period of fourteen years (from 1940 to 1953) have been scrutinised to determine frequency, breed, age and site incidence of the disease. A case was considered infected if a gross lesion had been recorded in the oesophagus, aorta, or in any other sites associated with the worms. Breed incidence has been ascertained in respect of those which were represented in sufficient numbers and the minimum number taken for this purpose was fifty in each breed. Many breeds uncommon in this area have been left out.

A brief review of the gross lesions noticed at the time of autopsy has been furnished. To study the microscopic features, sections of oesophagus and aorta from the site of the lesions were stained with Haematoxylin and eosin, Mallory's aniline blue and Mallory's phosphotungstic acid. Sections from the aorta were also stained for elastic tissue by Verhoeff's method (Lillee, 1948).

1. Incidence

Autopsy frequency: Table I given below furnishes information on the incidence of Spirocercosis for the eleven year period ending 1953.

Table I — Autopsy Incidence — 1943-53.

Year	Total No. of autopsies (dogs)	Spirocerca infection	
		Number	Percentage
1943	76	16	21.0
1944	86	13	15.0
1945	71	14	20.0
1946	93	16	17.0
1947	65	10	15.4
1948	77	13	17.0
1949	95	23	24.2
1950	93	24	28.8
1951	79	24	30.4
1952	72	29	40.0
1953	110	33	30.0

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From the above figures it will be seen that the overall incidence is 23.5% among 910 dogs examined post-mortem. This figure is far in excess of that recorded in the United States of America by Barron, Gulbert, False and Newberne whose figures are 17.65%, 20.0% and 5.4% respectively. Only in Sumatra does the infection appear to be much more common than in this country (40-50%).

Breed incidence:

The following table furnishes information in respect of the incidence in various common breeds of dogs brought for treatment in the College hospital. Incidence among other recognised breeds are not given for reason that they are not so numerous as to permit proper analysis.

Table II — Breed Incidence — 1940-53.

Breed	Number of dogs autopsies	Spirocerca cases	
		Number	Percentage
Alsatian	147	57	38.6
Terriers (of all types)	50	16	32.0
Dachsund	81	21	26.0
Spaniel	85	19	22.3
Nondescript ...	328	58	16.3

The above figures lend support to the popular contention among practitioners that Spirocerca infection is commoner in Alsatians than in other breeds.

Age incidence:

Table III gives information as to the approximate age of the dogs that showed spirocerca lesion at autopsy. Since the age of the dog was not mentioned in all cases, the total number represents only a portion of the material received during the period. The age recorded at the time of autopsy could not indicate the age of the lesions.

Table III — Age Incidence — 1943-53.

Age in years	Under 1 yrs.	1 yr.	2 yrs.	3 yrs.	4 yrs.	5 yrs.	6 yrs.	7 yrs.	8 yrs.	9 yrs.	10 yrs.	11 yrs.	12 yrs.	Over 12 yrs.	Total
No. of dogs affected	20	20	42	27	19	10	9	9	4	3	4	2	1	13	183
Percentage	10.9	10.9	22.75	14.8	10.35	5.5	4.95	4.95	2.2	1.7	2.2	1.1	0.55	7.15	

The above table shows that majority of cases were in age groups of 1 to 7 years. Twenty cases were below one year of age at the time of detection, and most of these showed nodular lesions on the adventitia of the aorta often with live worms inside.

2. Pathology

Frequency of gross lesions in various situations:

Table IV furnishes data on the site of occurrence of the lesion in 247 dogs of various breeds subjected to examination post mortem.

Table IV — Location of the Gross Lesions.

(a)	Pronounced thickening or nodule formation, single or multiple in the thoracic oesophagus alone	194	78.5
(b)	Nodule formation in stomach (and oesophagus, thoracic)	1	0.42
(c)	Arterial lesions (aneurism, atheroma, rupture) and oesophegeal nodule or nodules)	47	19.0
(d)	Arterial lesion alone	3	1.26
(e)	On the lumbar fascia over psoas magnum	1	0.42
(f)	Mediastinum, nodule	1	0.42

The above table shows that the most frequent site is the oesophagus and that in all cases where the oesophagus has been involved the arterial wall may not indicate any gross evidence of the disease. Failure to notice gross lesions in the aorta might be due to faulty examination of the intima at the time of autopsy. In old cases, the arterial lesions may have healed leaving only a minimal structural change not discernible on gross examinations. It was felt that cases with arterial lesions might be more in number than those recorded here.

Gross lesions:

In the thoracic oesophagus the lesions were present usually at a distance of about 1 to $1\frac{1}{2}''$ from the hiatus oesophigi. In occasional cases they might be as far forward as $4''$ from the cardia. Very often well-formed fibrous nodules, varying in number from one to four were observed at autopsy. The worms reach the oesophagus by migration from the aortic wall and initially set up chronic inflammatory reaction on the outside of the wall. There is induration and this progresses into a fibrous nodule which may reach the size of a marble. Occasionally they may attain the size of a hen's egg. These nodules initially outside, soon involve the wall and cause varying degrees of protrusion in the oesophegeal lumen. Simultaneously the worms burrow through the wall and open on the mucosal side, though occasionally this may not happen. The migration of the worms to the mucosal side leads to infection and subsequent suppuration. Such a nodule on section shows purulent areas separated imperfectly by thick fibrous septa. A large number of live worms may be found in the thick greenish yellow pus. Occasionally the worms wriggle out into the lumen and in one instance the number was 80. The escape of such a large number of worms into the oesophageal lumen might be a post-mortem phenomenon.

In one case the lesion in the form of a nodule of the size of a tamarind seed could be seen in the lesser curvature of the stomach, at a distance of about $1''$ from the pylorus. No worm could be detected in the nodule, nor was there any opening on the mucosa. Nodules with live worms were present in the thoracic oesophagus.

In four instances, the oesophageal nodule opened externally and ingesta escaped into the pleural cavity causing fatal septic pleuritis. In one such case the oesophageal

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nodule showed lateral communication into the aortic lumen and nearly 500 cc. of semi-clotted blood could be seen in the stomach and the intestinal contents were blood tinged.

In the case of a terrier male dog (A.34/43) which died of septic broncho-pneumonia, the oesophageal nodule communicated with the trachea at the level of its bifurcation. Small pieces of bone were seen in the small ulcerating tract which connected the nodule with the trachea. The trachea at that level was firmly adherent to the oesophagus and there was much induration around the sinus tract.

In 47 cases, gross changes could be seen in the aortic wall along with lesions in the oesophageal wall. In three instances, all in puppies, lesions were seen in the aorta only. The arterial lesions are thinning of the wall, pouching (aneurism), atheroma and rupture. Patchy atheromatous changes and thinning of the wall were observed more frequently than were aneurysm formation and rupture. In this series, six died of rupture of aneurism and haemorrhage into the pleural cavity. Initially the worms produce hypertrophy of the adventitia and small nodules on the outside and, in the early stages, live worms could be detected in these nodules. In the older lesions, no nodules were observed on the arterial wall but only thin-walled small saccular dilations.

In a large number of cases, there was thickening of the loose areolar tissue between the aorta and oesophagus and occasionally nodules containing live worms were present.

Another constant feature especially in long-standing cases was the presence of severe congestion of the middle mediastinum with diffuse minute red velvety granulation tissue.

In general it may be said that the infection produces haemorrhages, inflammatory reaction, necrosis and purulent streaks in the tissues through which the worm larvae migrate before they take up their final abode in the oesophagus. Much of these lesions resolve and only the residual effects are evident at autopsy. Pyaemic secondary infection of the kidneys was seen in five cases. In all these cases numerous small abscesses, evidently of haematogenous origin, were present in both kidneys. In four long-standing cases there was severe aplastic anaemia and in six cases anaemia of less severe degree. It is possible that the anaemic condition might have been brought about by the long-continued action of toxic substances, produced by the worm, on the haemo-poietic tissue.

Microscopic lesions:

Externally the oesophageal nodule showed severe fibrosis, which at places broke up to form sinus tracts and opened on the mucosa. The fibrous tissue of the capsule was prolonged into the interior of the nodule as septa. The sinus tracts were lined with cellular necrotic debris and clusters of pyogenic cocci. The contents of the suppurating areas of the nodule were made up of worms (cut in various fashions) necrotic tissues, pus cells and clusters of pyogenic cocci. Internal to the fibrous capsule and in the wall of the sinus tracts there was much granulation tissue. Dead and living fibrous tissue could be seen. The latter was supplied abundantly with blood vessels. The muscular coat of the oesophagus was considerably thinned and the fibres were severely fragmented. The mucous glands in the sub-mucosa at the site

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FIG. 1

The uriniferous tubule of a cow foetus, aged three months. The crystals of uric acid in the lumen. Magn. 500x.

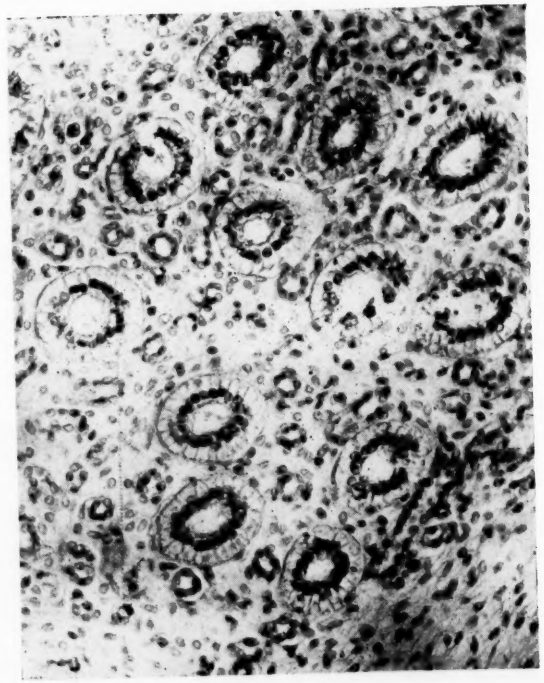

FIG. 2

A section of the renal tissue of a cow foetus, aged six months. In each tubule crystals of uric acid. Magn. 200x.

PLATE 1

(Article by S. Talanti on pages 374-376)

PLATE 1

FIG. 1
Oesophagus with Spirocerca lupi attached to the mucosa. Note the disappearance of the mucus glands under the site of attachment and necrotic areas in the wall (H.E.X14).

FIG. 2
Section of the aorta to show the immature w
(H.E.X14).

FIG. 3
Aorta showing severe intimal thickening (H.E.X23).

FIG. 4
Aorta showing fragmentation of elastic fibres (Verhoeff's Stain. X.14).

(Article by K. P. Chandrasekharon, G. A. Sastry and M. N. Menon on pages 388-395)

had degenerated completely and disappeared. Similar changes were observed in the stratified squamous epithelium in and around the place where the worms had made the opening (Fig. 1).

In the aorta, fibrous nodules showing cut ends of immature worms were seen in early cases (Fig. 2). This change was confined to the adventitia and the intima appeared to be more or less normal. Occasional eosinophils and round cells were present around the worms, but eosinophilic infiltration to the extent seen in many other parasitic infections was absent in the sections examined. This process of nodule formation was absent from the arterial adventitia in all the cases examined, probably because according to the life history of the worm, the arterial wall forms only a temporary abode of the parasite. It is possible that in mild infections the inflammatory reaction might resolve leaving behind some amount of scarring only. Where the infection of the wall was heavy, fibrous nodules, partially hyalinsed with calcified worms inside, could be seen. In the majority of cases examined histologically, one finds only the residual lesions left behind by the migrating immature worms. These changes include patchy fragmentation and loss of elastic fibres (demonstrated well by Verhoeff's stain) atrophy of the muscle fibres and their replacement by collagen fibres, patchy calcification in deeper portions of the intina, fibrous proliferation with partial hyalinisation in the intina, and denudation of the superficial endothelium (Figs 3 & 4). The pouching of the wall, a characteristic of old cases ,can be explained as being due to the loss of elastic and mulscular tissues. Though in many cases macroscopic signs of atheroma were evident this could not be substantiated histologically because of the absence of the lipoidal changes characteristic of this process. The aneurisms were frequently of the saccular type, but in the case of those that eventually ruptured, a dissecting type was noticed.

3. Diagnosis

In every case in which spirocerca infection has been detected at autopsy, no antimortem diagnosis by faecal examination was made. In view of the great frequency of the condition in this country, special search was made for eggs of *S. lupi* at every faecal examination. The causes for the failure to detect every positive case may fall under any of the following heads:

(1) the disease was not suspected by the clinicians and no faecal examination was made.

(2) the lesion in the oesophagus may be in the early stages and the worms have not burrowed sufficiently to enable the eggs to be passed into the alimentary tract.

(3) intermittent deposition of the eggs by the female worm precluding detection of all positive cases at a single faecal test.

4. Discussion

The survey is fairly comprehensive and representative of the common breeds of dogs that frequent the clinics in this city. It must be noted that the figures represent the autopsy incidence only and that the actual incidence might be much more. The higher incidence in Alsatians as compared with other breeds, might possibly be related to some habits peculiar to this breed and not due to any breed susceptibility. As regards age incidence, though a large percentage of cases were noticed in age groups of 1-7 years, it was significant that in 20 dogs the disease was observed at ages between

4 to 10 months. The lowest age at which the disease was noticed was 4 months. False and Neweberne (*loc cit*) recorded the lowest age of infected dog as 5½ months. They stated that this was possible because of the extended period of migration before adults appear in the oesophagus.

From a perusal of the table showing location of the gross lesions one gains the impression that concomitant with the oesophageal lesion, changes in the arterial wall are not common. This probably may not be quite true, for many cases of arterial lesion might have been overlooked. One cannot see evidence of gross lesions on the aorta always, because it forms only a temporary abode during the journey of the worm larvae to the oesophagus. Lesions set up initially might have healed leaving only microscopic evidence of the original infection. The lumbar fascia and mediastinum are among the rare sites for the localisation of the worm. The severe congestion of the middle mediastinum and formation of red velvety granulation tissue noticed by us, do not appear to have been mentioned by other workers.

The manner in which the pyaemic lesions of the kidneys and anaemia could be brought about in spirocercosis has been indicated. It should be emphasised that the aetiologic association of spirocercosis with these two conditions has been firmly established.

Seibald *et al* (1955) reported ten cases of malignant oesophageal tumours in the dog and in nine of them evidence of spirocerca lupi infection, either direct or indirect was shown. Comparative incidence of oesophageal neoplasms in the infected and the non-infected dogs showed a significantly higher percentage in respect of the former group. On this basis these workers believed that Spirocerca lupi might be an inciting factor in malignant oesophageal tumours. In none of the 910 autopsies, almost all of which were personally conducted by the senior author, could any evidence of tumour formation in relation to the worm lesion in the oesophagus be seen.

Summary

A survey is made of the Spirocerca lupi infection in respect of autopsy frequency, breed and age incidence. It was found that overall incidence among 910 dogs was 23.5%. Among the breeds common in this area, Alsatians showed the highest incidence (38.6%) and the non-descripts the least (16.3%). It was suggested that the high incidence in Alsatians might possibly be related to some peculiar habit of this breed and not due to any special breed susceptibility. The majority of the dogs with infections detected at autopsy were in age groups of 1 to 7 years and only 20 were aged below 1 year (4-10 months).

The location of the lesions in respect of 247 cases are tabulated. In all except 3 cases, oesophageal lesions were observed. The three cases which showed arterial lesions only were aged below one year. In 47 cases, the arterial wall in the vicinity of the infected oesophagus presented gross lesions. It was felt that more instances of arterial lesions might have been discovered, had a special search been made at the time of the autopsies. The nature of the lesion seen in the oesophagus and aorta did not differ from what has been described by other workers.

In four instances the nodules in the thoracic oesophagus ruptured externally causing septic pleurisy. In one instance the nodule communicated with the trachea at its bifurcation through a small sinus tract causing fatal broncho-pneumonia. In six

cases the weakened aortic wall ruptured resulting in haemorrhage into the pleural cavity and death. In many instances severe congestion of the mediastinal pleura and formation of diffuse velvety granulation tissue occurred. It is suggested that this might have been due to local venous obstruction caused by the enlarging nodules and to the irritating influence of the migrating worms.

In five cases of spirocerca infection pyaemic nephritis was present and it is suggested that septic foci in the oesophagus might probably be the primary site for the kidney infection.

Aplastic anaemia in five dogs and less severe types of anaemia in six dogs were noticed in long standing cases of the disease. It is possible that anaemia might have been set up in these cases by the action of some toxic substances liberated by the worm.

The arterial lesions, grossly described as atheroma, exhibited no lipoidal degeneration, but showed chiefly replacement of elastic tissue by collagen fibres, atrophy of the media and patchy intimal proliferation.

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CAUSES OF MORTALITY IN CHICKENS UP TO TEN DAYS OLD

by

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Introduction

A large proportion of the eggs set in any hatchery are not fertile, fail to hatch or the chickens, if hatched, die within a few days. The causes are diverse and vary from country to country. As the relative importance of different diseases in South Australia was not known it was decided to make an investigation into the general causes of death in very young chickens.

A hatchery was chosen which drew its eggs only from Government tested accredited pullorum free flocks and in which full records of all eggs, the supplier, the fertility and the hatchability were kept. Arrangements were made for a considerable number of the chickens hatched to be sent to a particular rearer whose premises could be visited daily except Sundays. All chickens sold to this farm were traced back to the supplier and every chicken which died was examined post mortem. The following information was then available on each batch of birds:—

1. Supplier,
2. Breed,
3. Hatchability,
4. Percentage of deaths in first 10 days of life,
5. Cause of such deaths.

Records were also available of the fertility and the degree of humidity at which the eggs were hatched. The fertility records paralleled the hatchability on a higher level and are not included. The degree of humidity was not found to affect the result as judged by the loss of chickens up to 10 days old and will not be considered further.

General Findings

In all 95,192 eggs were set of which 64,331 or 67.6% hatched. Of these 20,746 or approximately one third were traced through to 10 days old. The causes of death were found to be omphalitis, unabsorbed yolk sac, pneumonia, uricaemia and pullorum disease (despite the strict testing of all stock). As it was not possible to collect any birds dying on Sundays, a proportion of the carcases were too decomposed for examination. The total of these was divided between the different causes of death in the proportion each occurred in the undecomposed birds. The total so distributed was 14.6% of all the dead birds.

Where statistical analysis has been employed the decomposed birds have been omitted as they were too few to be divided between each batch.

Of the 20,746 birds 1,507 or 7.3% died during the first 10 days of life. The causes of death and their relative importance are shown in Table I.

TABLE I.
DEATHS IN CHICKEN ACCORDING TO BREED AND SUPPLIER

Group	Birds No.	Dead No.	Dead %	Omphalitis No.	Omphalitis %	Unabsorbed Yolk No.	Unabsorbed Yolk %	Pneumonia No.	Pneumonia %	Uricaemia No.	Uricaemia %	Pullorum No.	Pullorum %	Percentage of all deaths due to:— Omph.	Un. Yolk	Pneumonit	Uricaemia	Pull.
Australorp.																		
Producer A	1794	235	13.0	105	5.8	76	4.2	14	0.8	39	2.2	9	0.5	44.6	32.0	6.0	16.7	3.8
„ B	4274	416	9.8	193	4.5	150	3.6	42	1.0	24	0.6	8	0.2	46.3	36.0	10.0	5.8	1.9
„ C	1298	127	9.7	44	3.4	37	2.8	4	0.3	11	0.8	33	2.6	34.7	29.1	3.0	8.0	26.0
Total	7366	778	10.6	342	4.6	263	3.6	60	0.8	74	1.0	50	0.7	43.1	33.8	7.8	1.0	0.6
White Leghorn.																		
Producer E	1695	81	4.7	38	2.2	31	1.8	7	0.4	5	0.3	1	—	46.9	38.2	8.6	6.1	1.2
„ D	2068	165	7.9	108	5.3	52	2.5	4	0.2	0	0.0	1	—	65.4	31.5	2.4	0.0	0.6
„ G	1055	79	7.4	55	5.2	22	2.0	1	—	1	—	0	0.0	69.6	27.8	1.3	1.3	0.0
„ H	572	40	7.0	27	4.7	9	1.5	1	—	1	—	1	—	67.5	22.5	2.5	2.5	2.5
Minor	506	39	7.7	25	4.9	12	2.4	0	0.0	0	—	2	0.3	64.1	30.8	0.0	0.0	5.1
Total	5896	404	6.8	253	4.3	126	2.1	13	0.2	7	0.1	5	0.1	62.6	31.2	3.3	1.8	1.2
Cross Bred.																		
Producer A	2221	128	5.7	58	2.6	40	1.8	17	0.7	12	0.5	1	—	45.3	31.3	13.3	9.4	0.8
„ G	2422	78	3.2	48	2.0	13	0.5	1	—	12	0.5	7	0.3	61.5	16.0	1.0	16.0	10.2
„ F	621	22	3.6	12	1.9	6	1.0	1	—	3	0.5	0	0.0	54.5	27.3	4.5	13.5	0.0
Total	5264	228	4.4	118	2.2	59	1.1	19	0.4	27	0.5	8	0.2	51.7	25.9	8.3	11.9	3.5
Rhode Island.																		
Total (A)	2220	89	3.9	44	2.0	22	1.0	12	0.5	6	0.3	5	0.2	49.4	24.7	13.4	6.8	5.6
All Breeds.																		
Total	20746	149	7.2	757	3.7	470	2.3	104	0.5	114	0.5	68	0.3	50.5	31.4	6.9	7.6	4.5

From this it will be noted that half the deaths are due to omphalitis, with unabsorbed yolk sacs accounting for the greater proportion of the other deaths. The overall death rate of 7.2% is not high and indicates that the method of rearing was satisfactory. Pullorum disease probably can be eliminated in due course as the flocks are tested repeatedly over the years. All in this series but a very few odd cases occurred in two successive batches from one producer. Uricaemia was considered to be due probably to Vitamin A deficiency as it occurred only with certain suppliers and varied with the supply of greenfood they had available. It could, have been due also to excess of animal protein (Patterson, 1928) or to a lack of this material (Bird, Rubin, Whitson and Haymes, 1946) but no evidence of either could be found. It was more noticeable in the heavy breeds than the White Leghorns but this may have been due to the different sources of supply. These two diseases—pullorum disease and vitamin deficiency—together responsible for 12.4% of all deaths, are avoidable.

Pneumonia

The third small group of deaths is that due to pneumonia. This was considered to be the cause when areas of pneumonic consolidation had occurred and streptococci were isolated even if the areas were small in extent. These may have been due to chilling but if so, there was no obvious reason, as all the chickens were treated alike. However, there was some difference between suppliers and between batches. The tendency was for losses to occur sporadically but usually occurred in the chickens from the same supplier. Thus crossbreds were almost entirely supplied by two producers A and G. Table II compares the results obtained from birds from each producer. With birds from A pneumonia occurred on five occasions whereas with G's birds it was only encountered once and then only in one chicken. Statistically this is significant.

TABLE II.

PNEUMONIA — CROSS BRED CHICKEN

Producer		Batch No.									
		1	2	3	4	5	6	7	8	9	10
A	No. of chicks	474	204	75	136	275	127	307	189	180	254
	% Pneumonia	0.8	0.0	2.7	0.7	1.8	0.0	1.0	0.0	0.0	0.0
G	No. of chicks	116	102	132	97	123	102	156	102	107	205
	% Pneumonia	0.0	0.0	0.0	0.0	0.0	0.0	0.6	0.0	0.0	0.0

It also appears that here was a variation between the breeds. Then the figures for Australorps and White Leghorns are compared proportionately three times as many Australorps as White Leghorns died from pneumonia — 59 Australorps (0.78%) to 13 White Leghorns (0.25%). When analysed statistically this difference is highly significant, but this must be treated with some reserve as the sources of the various breeds were different. However, unlike the crossbreds, analysis of the results from each breeder within each breed failed to show any significant differences between breeders. Although the same breeders did not supply both Australorps and Leghorns, it is very probable that the difference shown is significant and that Australorps were more susceptible to pneumonia than White Leghorns.

The cause of this greater susceptibility in Australorps may have been due to any of several factors other than the breed itself. For example, many Australorps were raised from eggs laid by pullets in their first season, while nearly all White Leghorns were from eggs laid by hens in their second or third seasons. The effect of this and other factors cannot be assessed at present, and we merely record the fact that our figures show that under the condition of the investigation Australorp chicks were more susceptible than White Leghorns.

In both breeds the number of birds dying from pneumonia decreased as the season advanced. This decrease was more marked in Australorps than in White Leghorns and may have been due to climatic conditions. However, they were in adequately heated brooders within a building, and outside temperatures and conditions should not have had any direct effect on them except possibly the degree of humidity.

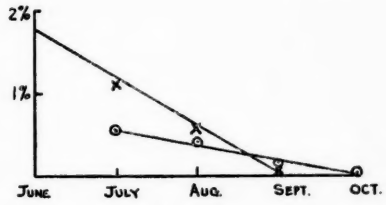

FIG. 1
Deaths from Pneumonia
Australorps x————x
White Leghorns ⊙——⊙

The figures are shown in Figure I. In both cases the drop is approximately linear, and if the number of deaths for June-July is compared statistically with that of August-September the difference is significant ($p = 0.02$).

Omphalitis and Unabsorbed Yolk

Omphalitis, the main cause of all deaths, was recorded if the yolk sac was unabsorbed and had become infected; if not infected the case was recorded as "unabsorbed yolk sac". Any cases of peritonitis were also regarded as due to omphalitis, whether or not the yolk sac had been absorbed. Thus on term "omphalitis" would probably be more correctly designated as yolk sac peritonitis but this is cumbersome. Although in the cases recorded as unabsorbed yolk this material did not look infected—the whole post-mortem appearance being of a "sterile" lesion—and although we endeavoured to culture organisms, there is in fact no proof that these were not due to bacterial or viral infection with some organisms which did not yield growths under the conditions of the study. These two related conditions together comprised the major proportion of deaths, 81.9% of all deaths being due to them (Table I).

It is widely held that whether or not the yolk sac is absorbed is affected by hatchery conditions, but in the present investigation hatchery conditions such as humidity and small variations in hatching temperatures did not appear to affect the incidence. Although this incidence varied greatly from batch to batch, there was a marked difference between Australorps and the other breeds. When the percentage incidence in each batch is plotted graphically, as in Figure II, it is seen that in the majority of White Leghorns, Rhode Island Reds and crossbred chickens the percentage death rate was under 2%, whereas in Australorps the percentage death rate was

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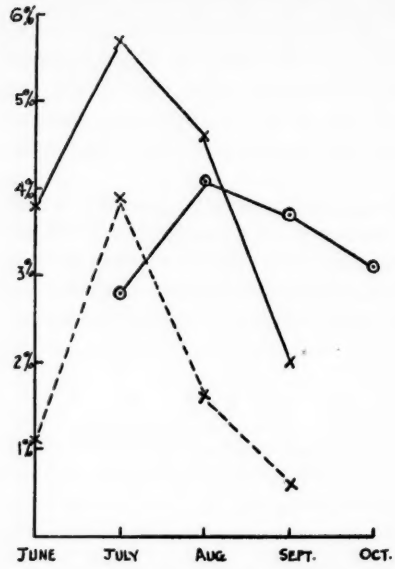

FIG. 3
Deaths from Omphalitis
Australorps x————x
White Leghorns o————o
Rhode Island x-----x

greater than 2% in over half the batches and even reaches 10%. It is, of course, possible that this apparent breed difference is due to suppliers rather than to the breed, but chickens from one producer occurred in three different breeds, Australorps, Rhode Island Reds and crossbreds. His birds gave results similar to the breed average for each breed, and it would therefore appear that a true breed differentiation occurs.

The percentage of the total deaths which were due to infection of the yolk sac or peritoneal cavity, referred to here as omphalitis, varied according to breed and supplier. This was true whatever the bacterial cause of the condition, though *Escherichia coli* was present in nearly every case either alone (in 85%) or with streptococci (in 8%) or proteus (in 3%). Streptococci were present, either alone or in combination with coli, in 10% and with proteus similarly in 5%. In one case *Pseudomonas pyocyanea* was the only organism recovered. Table I which gives all causes of deaths shows that 43% of the deaths in Australorps were due to this disease against 61% in the White Leghorns. As, however, more Australorps died than White Leghorns the figures for the two breeds are almost identical when calculated as a percentage of the total birds hatched—4.6% for Australorps and 4.3% for White Leghorns. These figures are interesting, as they indicate that the 3.6% higher death rate in Australorps was entirely due to a greater number of deaths in the minor causes of death and not omphalitis.

A further point of interest is that all batches of Australorp eggs were fumigated after 18 days incubation with formalin at a strength of 35 ml. per 100 cubic ft. incubator space, whereas almost all the White Leghorns were not. The hatching incubators were fumigated weekly when Australorp eggs were present, and thus there would have been little build up of infection in the incubators themselves. Apparently fumigation of the actual eggs was not having any practical result in reducing omphalitis,

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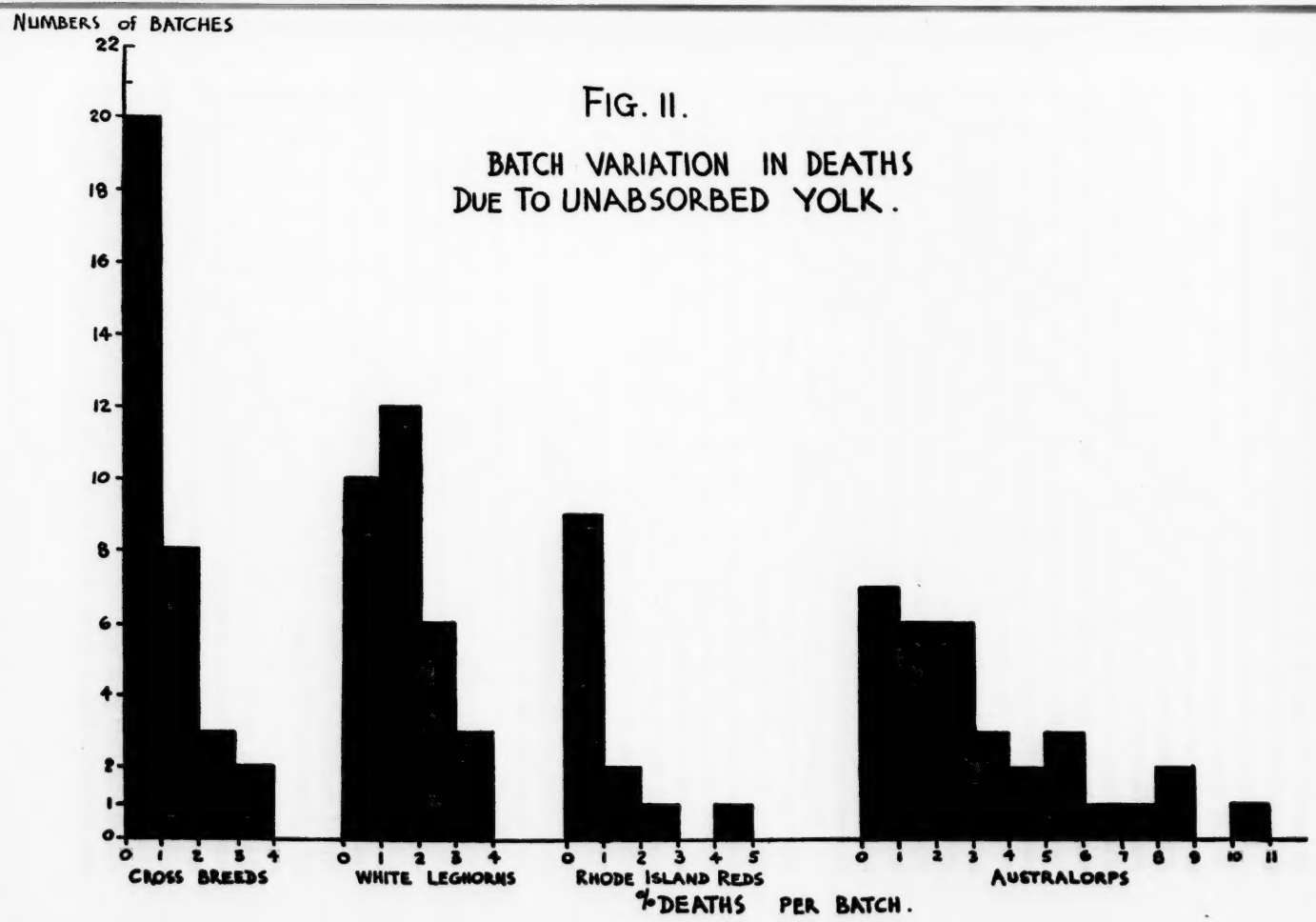
FIG. II.

BATCH VARIATION IN DEATHS
DUE TO UNABSORBED YOLK.



or possibly the strength used was insufficient. Whatever the reason, whether or not the eggs themselves were fumigated had no effect on the incidence of omphalitis.

As with pneumonia, the incidence of omphalitis varied with the month. However, instead of falling steadily as in pneumonia, the peak incidence was on the second month of hatching of each particular breed. Figure III shows the results for Australorps, White Leghorns and Rhode Island Reds (these latter have not been included in the pneumonia figures, as too few died). The Australorps and Rhode Island Red chicken started hatching in June and continued till September, while the White Leghorns started in July and finished in October. The figures for each breed show a high incidence of deaths in the second month and a steady fall in the next two. This result was a little unexpected, as it had been thought that there would be a build up of infection as the season proceeded. It seems unlikely that seasonal conditions leading to soiling of the eggs by infected earth could have been directly responsible, as all the breeding hens were kept under the intensive system and no soiling of the eggs from wet should have occurred. Another possibility is that the incubators and hatchers were more crowded during the second month than at other periods, i.e. that more chickens were hatched in the second month. However, the figures show that this was not so, as 14,369 Australorps were hatched in August, but only 8,107 in July.

The figures for Leghorns showed the same tendency to a lesser degree. However, there was a number of producers, and variations occurred between these producers, making the results less clear cut. Thus chickens from producers H and G showed the same tendency as the Australorps. Those from producer P gave a steady high figure throughout the year, while those from producer E showed a drop in the middle, with peaks at the beginning and end of the season. Obviously the figures for any particular time depended to a large extent on the proportion of chickens available from each producer, and this varied considerably.

TABLE III.
OMPHALITIS — WHITE LEGHORNS

Producer	Percentage Omphalitis in Successive Batches										
D	9.3	2.0	3.9	1.9	3.0	3.9	4.9	4.4	3.9	5.2	1.0
E	1.3	2.9	0.0	0.0	1.9	1.6	1.0	1.7	3.3	2.3	1.9
G	2.0	6.8	4.5	2.2	0.9	1.1	2.2	3.3	4.4	5.5	8.8

In this breed it was possible to distinguish variations between the producers in the amount of omphalitis. Producer E consistently produced chickens with a low rate of omphalitis. In Table III the death rate from this disease in successive batches of chickens from Producers D and E is shown. Quite clearly those of Producer E regularly had a lower rate. The incidence of this disease was therefore in part due to the producer, but no obvious reason for this difference could be found, although it was noted that Producer E kept all his breeding stock in pens of 12 with ample nesting space, while all other producers had large units of several hundred birds. It is difficult to see, however, how this could have affected the death rate from omphalitis in the resultant chicken, particularly as a noticeable feature of the deaths from this disease was their great variation in number in different batches of chickens from any one producer. This is shown clearly in the table and it occurred in other breeds also.

Thus in Rhode Island Red chickens, when all came from one producer and so far as could be ascertained every batch was treated identically, successive batches showed the following percentage losses from omphalitis:—1.0, 1.3, 4.5, 1.9, 1.0, 6.8, 1.9, 1.4, 1.5, 1.0, 0.9, 0.0, 0.0. Not only was there this variation between batches of any one producer's chickens, but a high incidence in the chickens from one producer in any one hatch did not by any means indicate that there would be a high incidence in the chickens from another producer in the same hatch, i.e. although hatched at the same time under identical conditions there was great variation between the death rates of chickens from different producers. There was no constancy as to which producer's chickens had the highest rate. Sometimes it was one, sometimes another.

This evidence indicates that some factor other than the source of origin, the treatment of eggs before setting and the conditions in the hatchery affects the incidence of omphalitis. In order to be certain, all chickens could be identified as to source, each producer's chickens were transported to the rearing quarters in separate boxes and were reared in separate 100 birds compartments of the brooders. It may be that the conditions of transport or the conditions in the separate compartments of the brooder varied sufficiently to affect the incidence of the disease. It was noticeable that the highest peaks of incidence occurred during July though not necessarily in the same batches, which is difficult to attribute to the influence of any factor which would be operating throughout the season. Whatever the reason, the significant fact was that the incidence of omphalitis lessened as the season progressed.

In all breeds it was interesting to note that the proportion of deaths due to omphalitis fell in each case between the first and last months by almost identical percentages despite the fact that the actual total of deaths varied between the breeds. Thus the total number of deaths in White Leghorns rose from 5.1% to 9.4%, while it fell slightly in the case of the other two pure breeds. But in all breeds the proportion of deaths due to omphalitis fell, by 42.3% for Australorps, 42.8% for Rhode Island Reds and 40.5% for White Leghorns. On this occasion therefore, as the season progressed the losses from omphalitis decreased, whether the total death rate rose or not, and this decrease was a constant proportion of the amount of omphalitis present at the beginning of the season whatever the breed.

Whatever factor or combination of factors was operating to produce this drop in the death rate from omphalitis in the later months it was not operative, at least so far as White Leghorns were concerned, on other causes of death. In Table IV it will be seen that in White Leghorns the percentage death rate from all causes rose from 5.1% to 9.4% between July and October, whereas deaths from omphalitis decreased and the percentage of total deaths due to this disease fell from 55.1% to 32.8% i.e. the increased death rate as the season advanced was due to causes other than omphalitis.

Discussion

Although the work only revealed clear evidence of significant variations between one producer and the others in the case of omphalitis itself, it did show the great importance of the omphalitis-unabsorbed yolk complex even when eggs from well managed farms are concerned. It is possible that the complex is one problem and that whether or not infection occurs is fortuitous. It bears no relationship to the type

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DEATHS FROM OMPHALITIS RELATED TO TIME
TABLE IV.

Date	Total Hatch	No. birds in experiment	No. Deaths	% Deaths	Deaths due to omphalitis No.	Deaths due to omphalitis %	% omphalitis in total deaths	Decrease in % of deaths due to omphalitis (1st to 4th months)
					Australorps			
June	4859	714	63	8.8	27	3.8	42.8	
July	8107	1875	246	13.1	107	5.7	43.4	
Aug.	14369	2611	286	11.0	118	4.6	41.2	
Sept.	14100	2166	174	8.0	43	2.0	24.7	42.3
					Rhode Island Reds			
June	1898	612	17	2.8	7	1.1	41.2	
July	2500	612	43	7.0	24	3.9	55.8	
Aug.	2349	306	11	3.6	5	1.6	45.5	
Sept.	1271	640	17	2.6	4	0.6	23.5	42.8
					White Leghorns			
July	9850	1925	98	5.1	54	2.8	55.1	
Aug.	15526	1233	96	7.8	50	4.1	52.0	
Sept.	17091	1518	124	8.1	57	3.7	45.9	
Oct.	12713	714	67	9.4	22	3.1	32.8	40.5

of "hatch" as recorded by the hatcheryman. A good "well finished" hatch was just as likely to have as high a proportion of deaths from omphalitis as one recorded as bad. The percentage of eggs hatching did not affect the numbers of deaths from omphalitis. Any particular batch could have a high percentage hatch with either a high or a low death rate in the resultant chickens. In fact the survival rate at 10 days, when regarded as a percentage of the number of eggs set, was more affected by the number of chickens hatched than by the number dying after hatching. The figures for the major producers are shown in Table V. For instance chickens from producer E

TABLE V.

SURVIVAL RATE RELATED TO PRODUCER AND TO THE NUMBER OF EGGS SET

Breed	Producer	No. Eggs Set	% hatched	% of chicken hatched which died within 10 days	% of chicken surviving of eggs set
Australorp	A	9476	65.7	13.0	57
	B	20139	66.9	9.8	60
	C	5080	66.3	9.7	60
Leghorn	D	6815	77.7	7.9	72
	H	1489	72.3	7.0	67
	G	7459	62.3	7.4	58
	E	11400	69.0	4.7	66
Cross Breed	A	5302	67.4	5.7	64
	G	10755	61.7	3.2	60
Rhode Island Reds	A	19838	69.4	3.9	67

had a low death rate after hatching. It was only little over half that of producer D, (4.7% against 7.9%). Nevertheless 72% of producer D's eggs produced chickens alive 10 days after hatching whereas producer E's eggs showed only 66% of eggs producing chicks alive at 10 days. Again in crossbreds 64% of all producer A's eggs produced chickens alive at 10 days although the death rate after hatching was 5.7% (Table I). Only 60% of producer G's eggs had similarly produced chicks living to 10 days despite a lower death rate after hatching (3.2). This was due to the bad hatching rate of eggs from producers whose chickens showed low percentage death rates.

This work was done at a hatchery where all care is taken. All farms supplying fertile eggs are visited and inspected by the hatchery owner and probably the eggs supplied are produced under better than average conditions in the hatchery and brooder house were, so far as could be ascertained at least as good as the average, while the time elapsing between sexing and arrival at the brooder was negligible. The chickens could therefore be considered to be exposed to unfavourable influences for less than is normally the case under commercial conditions. Nevertheless the incidence of disease, particularly omphalitis, was as high as is normally encountered under less favourable commercial conditions. It was particularly noticeable that although every batch appeared to be subjected to identical conditions, variations between batches was considerable. Even batches of several hundred when split into hundreds showed vastly different death rates between the separate loss of hundreds. This could not be related to the brooder, as it occurred indiscriminately in any brooder. The variations made statistical analysis difficult, and several observations thought to

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be significant were found to be non-significant statistically. Taken as a whole, however, the results failed to demonstrate any specific factor influencing the incidence of omphalitis or of unabsorbed yolk, although there was a graded difference between producers, and in White Leghorns one producer could be demonstrated to have significantly less omphalitis in his chickens than the other producers. If Pearson (1951) is correct in his suggestion that omphalitis is mainly due to infection of the eggs by the hen during production, this could be explained by the assumption that the producer had less infected birds, and as noted above, he was the only one who kept his birds in small units. This would render identification of birds with "vent gleet" for example, relatively easy and would tend to confirm Pearson's suggestion. In this case, however, it was recorded that this producer's chickens were large and considered easy to sex, whereas the chickens from the producer whose birds showed the highest incidence of omphalitis were reputed to be very difficult to sex. As only one sexer is employed at any hatchery, it was not possible to demonstrate whether or not the personal element influenced the amount of injury received by the chicks during sexing. Neither was it possible to determine whether the act itself was inimical to the birds. This was because so few chickens are now sold unsexed that no large enough group of such chickens could be obtained. However, the observed facts can be explained readily if the act of sexing is considered to influence the result. The random occurrence of high rates of incidence in odd batches can be ascribed to personal factors affecting the sexer, and the higher incidence in the earlier part of the season with the steady fall as the season progressed to increased skill in handling the chickens. This conception appears with the statement of Harry (1957) that yolk sac peritonitis is connected with handling by the sexer.

The work failed to reveal definitely any particular factor or factors which could be incriminated as affecting the death rate in young chickens under reasonably satisfactory conditions of egg production, incubation, hatching and brooding, although the death rate was considered fairly high. It is likely that this rate was affected, in this series, mainly by extraneous factors working on odd batches and not by general hatchery conditions. The majority of the deaths in all batches occurred around the 6th day of life. This was because 71% of all deaths from omphalitis and 17% of all deaths from unabsorbed yolk occurred between the 4th and 7th days while 65% of all cases of pneumonia were recorded between the 5th and 7th days. Early deaths on the 2nd and 3rd day were usually due to uricaemia, as 84% of all deaths from this cause occurred on the 2nd and 3rd days of life.

In this investigation, the number of chickens dying after hatching did not affect the overall ratio of chicks at 10 days/eggs set so greatly as did the number actually hatched. Under the conditions of this experiment the causes of low fertility and low hatchability were of greater economic importance than those of disease in the hatched chick during its early post-hatching life.

Summary

All deaths occurring during the first 10 days of life in 20,746 day old chickens hatched and maintained under identical conditions were investigated and the cause ascertained. The greatest single factor was omphalitis (48.7%) followed by unabsorbed yolk sac (30.7%). Minor causes of death were pneumonia (6.9%) uricaemia (7.8%)

and pullorum disease (4.6%). Pneumonia was shown to vary with the producer and the month of hatching, decreasing as the season proceeded. Omphalitis also varied with the time of hatching, being greatest in all breeds during the earlier part of the season. Whether or not the total death rate in a breed rose or fell as the season proceeded, the proportion of deaths due to omphalitis fell by 40% between the beginning and end of the season. In this series there was only significant evidence in one case that omphalitis in the chicken was affected by the farm of origin of the eggs, but as all were produced under first class commercial conditions it was not possible to study the effect of faulty farm management.

Acknowledgments

This work would not have been possible without the complete co-operation of Mr. E. O. Dorney of Dorney Hatchery and Mr. H. J. McCarthy of Dorney Poultry Farm. All statistical analysis was either carried out or checked by Mr. G. Wilkinson of the C.S.I.R.O., Department of Mathematical Statistics for whose assistance we are very grateful.

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REVIEWS

VETERINARY OPHTHALMOLOGY, by R. H. Smythe. Pp. VIII + 379 + 15 plates. (2nd Edition 1958. 42s. net). Bailliere, Tindall & Cox Ltd., London.

The author and publishers are to be congratulated on the production of a second edition, due to demand, after an interval of less than two years.

New diagrams and recent extracts from literature have increased the size by 21 pages. Several of the plates are different from those in the first edition, and there has been a general improvement in the method of presentation. The references to the literature are now at the end of each chapter. The book is in two parts and the first 138 pages are again concerned with the anatomy and physiology of the eyes of various animals.

The second part of the book is devoted to clinical ophthalmology. Congenital abnormalities are now dealt with as a chapter in this section and follow that dealing with the clinical examination of the eye. Clearer titles have considerably improved this part of the book.

The surgical references are still somewhat disappointing and no method of extirpation of the eyeball which might give promise of first intention healing is described.

The descriptions of entropion and ectropion are still too elaborate, but now the removal of the crystalline lens is described adequately.

Despite these few criticisms this book contains a wealth of information which can be studied with profit by all who are interested in advancing their knowledge of veterinary ophthalmology. The text of this edition, like the first, is presented well, and easy to read.

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DISEASES OF DOMESTIC ANIMALS IN NEW ZEALAND. Technical Committee N.Z. Veterinary Association, Inc. Wellington, N.Z. Price 35/-.

A VETERINARY surgeon who proposes to emigrate to New Zealand need not fear that there will be any lack of demand for his service. According to this book there are about 170 "significant" diseases of the domestic animals in that country. No doubt there are many conditions also which have not attained to the status of "significant". The diseases are grouped under headings Bacterial, Viral, Protozoal, Fungal, Parasitic, Nutritional, Metabolic, diseases due to poisons and Miscellaneous.

In each section the diseases are listed in alphabetical order which makes reference very easy. The information concerning each disease is displayed in concise form and related to a series of headings, i.e. species affected, incidence, aetiology, signs, pathology prognosis, treatment etc. The format is reminiscent of the "Aids series" of text books which are such a boon to medical and veterinary students because they give all the relevant information in a simple, useful and compact form.

This book is not one which is designed for the armchair reader but rather for a busy man who wants a book of reference to which he can apply for up to date essential factual information which is displayed concisely and without waste of words. There can be no doubt that it will be very useful to the "new-comers" to New Zealand and to veterinary students who are reading for their final examination.

There is a useful index. The table of contents lists a section containing eight appendixes, Veterinary Surgeon's Act, Dangerous Drugs Act, etc. This section is absent from the reviewers copy.

NEWS

Major R. C. G. Hancock, B.Sc., M.R.C.V.S., who retired in June from the post of Chief Veterinary Officer to the R.S.P.C.A., has been appointed to the staff of Aspro-Nicholas Limited. Major Hancock will be mainly engaged in organising and following up field trials of the Cobalt Bullet now in progress in Great Britain and overseas expects to hand over to his permanent successor in the course of the next few months.

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